The Honorable Ricardo S. Martinez, Chief Judge

UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF WASHINGTON
AT SEATTLE

SAMIT PATEL, individually and on behalf of all others similarly situated, Plaintiff, v. SEATTLE GENETICS, INC., CLAY B. SIEGALL, TODD E. SIMPSON, and JONATHAN DRACHMAN, Defendants.	No. 2:17-cv-00041-RSM **CONSOLIDATED SECOND AMENDED COMPLAINT — CLASS ACTION — FOR VIOLATION OF FEDERAL SECURITIES LAWS** Jury Trial Demanded

Lead Plaintiff Carl Johnson ("Lead Plaintiff"), individually and on behalf of all other persons similarly situated, by his undersigned attorneys, for his Consolidated Second Amended Complaint against Seattle Genetics, Inc. ("Seattle Genetics" or the "Company"), Clay B. Siegall ("Siegall"), Todd E. Simpson ("Simpson"), and Jonathan Drachman ("Drachman") (Siegall, Simpson, and Drachman are referred to as the "Individual Defendants"; Seattle Genetics and the Individual Defendants collectively are referred to as the "Defendants"), alleges the following based upon personal knowledge as to Lead Plaintiff and his own acts, and upon information and

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belief as to all other matters, based upon, *inter alia*, the independent investigation conducted by and through his attorneys, which included, among other things, a review of the Defendants' public documents, United States Securities and Exchange Commission ("SEC") filings, wire and press releases published by, and regarding, Seattle Genetics, conference calls and announcements made by Defendants, economic analysis of Seattle Genetics' stock price movement and pricing volume data, analysts' reports and advisories about the Company, private investigation, and information readily obtainable on the internet. Lead Plaintiff believes that substantial evidentiary support will exist for the allegations set forth herein after a reasonable opportunity for discovery.

NATURE OF THE ACTION

1. This is a class action on behalf of persons or entities, other than Defendants, who purchased or otherwise acquired Seattle Genetics' common stock between October 27, 2016 and December 27, 2016, both dates inclusive (the "Class Period"), seeking to recover damages caused by Defendants' violations of the federal securities laws and to pursue remedies under §§ 10(b) and 20(a) of the Securities Exchange Act of 1934 ("Exchange Act").

2. Seattle Genetics is a development stage biopharmaceutical company traded on the NASDAQ exchange under the symbol "SGEN." This case arises out of misrepresentations and omissions that Defendants made during the Class Period related to known liver toxicity of one of Seattle Genetics' most important drug candidates, SGN-CD33A, also known as Vadastuximab Talirine. Defendants spoke about SGN-CD33A regularly in conference calls with investors throughout the Class Period.

3. SGN-CD33A is a type of cancer treatment known as an antibody-drug conjugate ("ADC"). ADCs are a drug technology that uses antibodies to target specific antigens on the surface of cancerous cells, and delivers locally strong anticancer agents that would be too toxic to

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administer otherwise. Seattle Genetics' trials of SGN-CD33A focused on developing the drug to treat a type of blood cancer called Acute Myeloid Leukemia ("AML").

4. SGN-CD33A is the successor to earlier ADCs developed by Pfizer and Seattle Genetics.

5. Pfizer developed an ADC known as Mylotarg (Gemtuzumab ozogamicin). Mylotarg was manufactured and marketed by Pfizer from 2000 to 2010 as a treatment for AML. In June 2010, Pfizer withdrew Mylotarg from the market at the request of the FDA because an advanced stage clinical trial demonstrated that the fatal rate of treatment-related toxicity was significantly higher than standard chemotherapy with no corresponding benefit to cancer patients.[1]

6. Several years before the Class Period, Seattle Genetics began developing an ADC called SGN-33, also known as Lintuzumab. SGN-33 was a predecessor to SGN-CD33A. In September 2010, Seattle Genetics abandoned the clinical trial for SGN-33, and publicly stated that the clinical trial failed to meet its primary endpoint of extending overall survival. The Company did not provide any substantive details about SGN-33's failure, including whether safety issues were observed in the clinical trial.

7. Seattle Genetics repeatedly claimed that SGN-CD33A had a superior design and

[1] Besides the now-withdrawn approval for Mylotarg, the FDA has only approved two other ADCs: ADCETRIS (Brentuximab vedotin), developed by Seattle Genetics to treat relapsed or refractory Hodgkin lymphoma and systemic anaplastic large cell lymphoma, and Kadcyla (Trastuzumab emtansine), developed by Genentech to treat metastatic breast cancer. However, a Cowen & Company analyst has noted that Seattle Genetics' ability to rely on revenues generated from ADCETRIS is limited because that drug has largely captured its target market with limited potential for future growth. In addition, a Morning Star analyst has observed that the market for ADCs is fiercely competitive with ADC technology under development by Celldex Therapeutics, Immunogen, ADC Therapeutics and AstraZeneca, which directly compete with and threaten Seattle Genetics' market share.

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more advanced ADC technology than Mylotarg, allowing it to kill cancerous cells effectively without the toxicity that doomed the earlier drug. Specifically, throughout the Class Period, Defendants claimed SGN-CD33A did not share the toxic side effects of Mylotarg, and misleadingly touted the absence of liver disease in clinical trials, while omitting that internal information disseminated to Defendants and others within Seattle Genetics unquestionably demonstrated that SGN-CD33A was hepatotoxic.

8. Each of the Defendants was well aware throughout the Class Period that SGN-CD33A posed a high risk of liver toxicity (hepatotoxicity). Specifically:

a. Data from preclinical studies of SGN-33 confirmed hepatotoxicity. Several years before the Class Period, Seattle Genetics developed a predecessor ADC to SGN-CD33A known as SGN-33. Animal studies for this drug showed a high risk of hepatotoxicity, and Seattle Genetics ultimately abandoned the clinical trial because it failed to meet its primary endpoint of extending overall survival of patients.

b. Internal documents confirmed hepatotoxicity. Seattle Genetics maintained Safety Data Sheets for each of the drugs it developed, and the components used in its labs. For SGN-CD33A, the Safety Data Sheets confirmed high levels of hepatotoxicity. These Safety Data Sheets were widely distributed within the Company, and they were available to all employees on an intranet known as "The Linker."

c. A third-party risk assessment confirmed toxicity. According to a confidential witness who had direct access to the preclinical studies and the Safety Data Sheets, in the middle of 2016, Seattle Genetics procured a third party risk assessment, which concluded that the levels of toxicity associated with SGN-CD33A were unacceptably high.

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d. Contract manufacturer raised concerns about toxicity. Seattle Genetics relied on the Lonza Group ("Lonza"), a contract manufacturing organization ("CMO"), to help produce the payload and linker components of the ADC technology utilized in SGN-CD33A. After seeing the third-party risk assessment discussed above, Lonza suspended production of the components that it helped produce for SGN-CD33A.

e. Company reaction to CMO manufacturing suspension indicates its awareness of toxicity concerns. The Company was aware of both the CMO's suspension and the concerns. In response, the Company directed its health & safety engineer to attempt to deflect those concerns.

f. In-house toxicologist raised concerns. According to a confidential witness, Seattle Genetics' in-house toxicologist expressed concerns about the level of toxicity in SGN-CD33A, before being pressured not to do so.

g. Company rushed SGN-CD33A to clinical trials without fully understanding hepatotoxicity risks. According to another confidential witness, a Senior Scientist who worked extensively on the chemistry of SGN-CD33A and helped to synthesize the drug for human trials, SGN-CD33A was extremely toxic, and Seattle Genetics rushed the drug to clinical trials without fully understanding the risk of hepatotoxicity.

h. Individual Defendants knew Mylotarg and SGN-CD33A were similarly hepatotoxic. The Individual Defendants were micromanagers, who knew granular details about the toxicity associated with Mylotarg, and attended companywide meetings where deep concerns about hepatotoxicity associated with SGN-CD33A were expressed before the issuance of press releases.

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i. Individual Defendants' own statements before the Class Period show knowledge of hepatotoxicity risks. For years, Defendants Siegall and Drachman made statements to investors asserting familiarity with intimate details about the failure of Mylotarg and SGN-33, preclinical studies of SGN-CD33A, and the toxicity profile of SGN-CD33A.

j. Regulations required awareness. Seattle Genetics was required to make itself aware of, and report to the United States Food and Drug Administration ("FDA"), deaths and other serious adverse events in clinical trials. Under FDA regulations, sponsors conducting clinical trials are required to "promptly review all information relevant to the safety of the drug" and to "notify FDA of any unexpected fatal or life-threatening suspected adverse reaction as soon as possible but in no case later than 7 calendar days after the sponsor's initial receipt of the information." 21 U.S.C. §312.32. Accordingly, to fulfill regulatory reporting duties, Defendants had to have known and informed the FDA of the four deaths due to hepatotoxicity in SGN-CD33A clinical trials, and the six cases of observed liver toxicity.

k. Delayed reporting of SGN-CD33A adverse events suggests that serious adverse events were known at the time of Class Period statements. The small subset of SGN-CD33A serious adverse events that has been released to the public demonstrates that the average delay between the event date and the initial report of the event to the FDA was 41 days, and the median delay was 34 days. This suggests that even if the six hepatotoxic events were not reported until the final several weeks of the Class Period, they were known substantially earlier.

9. On December 27, 2016, the FDA placed a full clinical hold on the Company's

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Phase I/II trial of SGN-CD33A administered to stem cell transplant patients ("Stem Cell Phase I/II").

10. The FDA also placed partial clinical holds on two other Phase I trials of SGN-CD33A administered in combination with chemotherapy regimens in AML patients. The Company told investors that the trials subject to partial clinical holds would not enroll new patients, and that existing patients could continue to participate if they signed a revised consent form.

11. On this news, Seattle Genetics' stock price declined by $9.50 per share, or by over 15%, to close at $52.36 on December 27, 2016.

12. Like investors, analysts were stunned by Defendants' revelation. That same day, for example, Credit Suisse analyst Kennen McKay lowered the Company's price target by $10, and remarked that the announcement was surprising given that Defendants had created the false impression that SGN-CD33A had unique technology to "avoid the [toxicity] pitfalls" of Mylotarg.

13. On March 6, 2017, the Company announced that it would abandon entirely the Stem Cell Phase I/II trial and would adopt substantial risk mitigation measures to address hepatotoxicity in all other trials of SGN-CD33A. With these hepatotoxicity risk mitigation measures in place, the FDA lifted the partial clinical holds it had placed on two other Phase I trials.

14. On June 19, 2017, the Company announced in a press release that it would discontinue a Phase III trial for SGN-CD33A known as CASCADE because a high rate of death was observed in the trial due to unspecified "fatal infections." In this press release, the Company also announced its decision to suspend patient enrollment and treatment in all SGN-CD33A trials.

JURISDICTION AND VENUE

15. The claims asserted herein arise under Sections 10(b) and 20(a) of the Exchange Act (15 U.S.C. §§ 78j(b) and 78t(a)), and SEC Rule 10b-5 promulgated thereunder (17 C.F.R. §

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240.10b-5).

16. This Court has jurisdiction over the subject matter of this action pursuant to 28 U.S.C. §§ 1331 and 1337 and Section 27 of the Exchange Act (15 U.S.C. § 78aa).

17. Venue is proper in this District pursuant to Section 27 of the Exchange Act (15 U.S.C. § 78aa) and 28 U.S.C. § 1391(b) given that a significant portion of the Defendants' actions, and the subsequent damages, took place within this District. Seattle Genetics is a corporation incorporated in Delaware with its principal place of business in Bothell, Washington, within this District, and Defendants Siegall, Simpson and Drachman reside in or around Bothell, Washington.

18. In connection with the acts, conduct and other wrongs alleged in this Complaint, Defendants, directly or indirectly, used the means and instrumentalities of interstate commerce, including but not limited to, the United States mail, interstate telephone communications and the facilities of a national securities exchange.

PARTIES

19. Lead Plaintiff purchased Seattle Genetics' securities at artificially inflated prices during the Class Period as set forth in his previously filed certification (Dkt. No. 7-2), and suffered damages as a result of the disclosure of federal securities laws violations alleged herein.

20. Defendant Seattle Genetics is a Delaware corporation with its executive offices located at 21823 30th Drive, Suite 300 SE, Bothell, WA 98021. Seattle Genetics' shares trade on the NASDAQ national market system under the ticker symbol "SGEN."

21. Defendant Siegall is Seattle Genetics' co-founder, and at all relevant times, has been Seattle Genetics' President, Chief Executive Officer, and Chairman of the Company's board of directors.

22. Defendant Simpson became Seattle Genetics' Chief Financial Officer in October

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2005, and held that position at all times relevant hereto.

23. Defendant Drachman was appointed as Seattle Genetics' Chief Medical Officer and Executive Vice President, Research and Development in October 2013, and held that position at all times relevant hereto.

24. Defendants Siegall, Simpson, and Drachman are sometimes referred to herein as the "Individual Defendants."

BACKGROUND AND PRE-CLASS PERIOD EVENTS

Clinical Trials and Clinical Holds

25. A biopharmaceutical company generally conducts clinical trials in three phases. These phases are codified in FDA regulations.

26. Phase I studies "are designed to determine the metabolism and pharmacologic actions of the drug in humans, the side effects associated with increasing doses, and, if possible, to gain early evidence on effectiveness." 21 C.F.R. § 312.21. They are typically open label, which means that trial data is provided to, not blinded from, the sponsor.

27. Phase II studies are "typically well controlled" studies "conducted to evaluate the effectiveness of the drug for a particular indication or indications in patients with the disease or condition under study and to determine the common short-term side effects and risks associated with the drug." *Id.*

28. Phase III studies are expanded studies "performed after preliminary evidence suggesting effectiveness of the drug has been obtained, and are intended to gather the additional information about effectiveness and safety that is needed to evaluate the overall benefit-risk relationship of the drug and to provide an adequate basis for physician labeling. Phase III studies usually include from several hundred to several thousand subjects." *Id.*

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29. Occasionally, a sponsor will designate a single clinical trial as spanning two different phases of study (*e.g.*, a Phase I/II trial or a Phase II/III trial).

30. During clinical trials, sponsors are required to "promptly review all information relevant to the safety of the drug" and to "notify FDA of any unexpected fatal or life-threatening suspected adverse reaction as soon as possible but in no case later than 7 calendar days after the sponsor's initial receipt of the information." 21 U.S.C. § 312.32.

31. If the FDA determines that a trial protocol exposes subjects to unreasonable and significant risk, the FDA can halt or limit the trial by placing it on a total or partial "clinical hold." 21 C.F.R. § 312.42(b). FDA Regulations require the agency to inform the sponsor of a deficiency and attempt to resolve the matter ***before***[2] issuing a clinical hold, unless patients are exposed to a serious and immediate risk. 21 C.F.R. § 312.42(c).

Hepatotoxicity

32. Hepatotoxicity is defined as drug-induced liver injury by the FDA's Guidance for Industry on Drug-Induced Liver Injury: Premarketing Clinical Evaluation, available at www.fda.gov/downloads/Guidances/UCM174090.pdf. It is the most frequent single cause of safety-related drug marketing withdrawals in the last 50 years. *See Guidance for Industry on Drug-Induced Liver Injury: Premarketing Clinical Evaluation*, at 2.

33. According to the FDA, "[m]ost of the drugs withdrawn from the market for hepatotoxicity have caused death or transplantation at frequencies in the range of ≤1 per 10,000, so that a single cause of such an event rarely would be found even if several thousand subjects were studied. Several drug induced liver injury cases rarely have been seen in drug development programs of significantly hepatotoxic drugs." *Id.*; *Id.* at 14 ("The presence of even a single case of severe

[2] Emphasis has been added here and to all significant quotations in this complaint.

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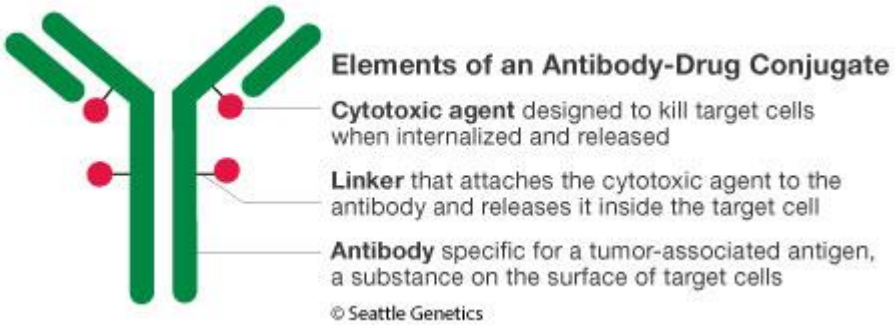
liver injury resulting from treatment in the premarketing clinical trials database is a signal of a high level of hepatotoxic risk."); *Id.* at 14-15 ("Experience has indicated that the occurrence of even one or two well-documented cases of this combination is ominous, indicating a likelihood that the drug will cause severe liver injury.").

34. The FDA's Guidance for Industry states that any serious case of liver toxicity must be handled as a serious adverse event, and promptly reported to the FDA even before a drug developer excludes other possible causes of the liver injury. *Id.* at 13.

ADCs and SGN-CD33A

35. Defendant Seattle Genetics is a clinical-stage biotechnology company that focuses on the use of ADCs to treat cancer. ADCs aim to harness antibodies to target delivery of toxic payloads to cancer cells while simultaneously seek to spare healthy cells susceptible to damage in non-targeted treatments like chemotherapy and radiation. Because ADC's are targeted and, aim to deliver the payload only where needed, they are considered to be a means of delivering more potent payloads to cancerous regions than could be delivered systemically.

36. The following illustrations show the elements of an ADC and the targeted delivery of a toxic payload:



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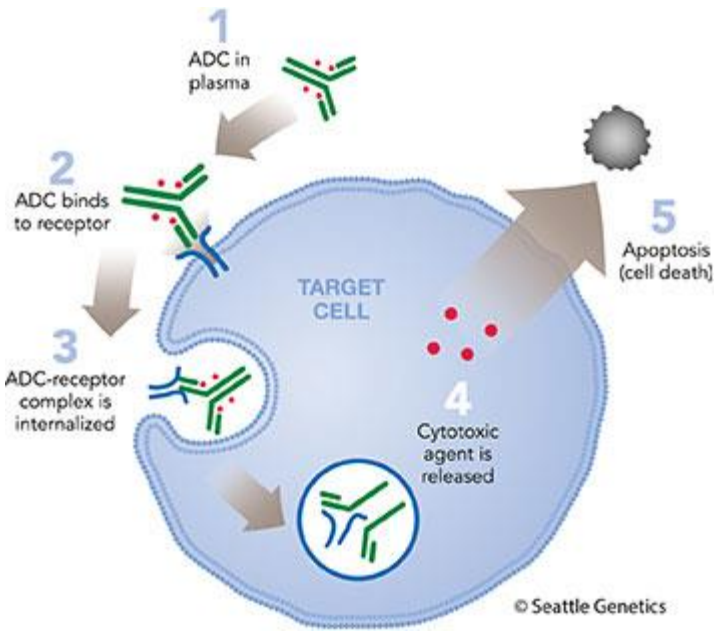

37. Specifically, SGN-CD33A uses the following components:

a. Cytotoxic agent (payload): SGN-CD33A uses highly toxic payloads known as pyrrolobenzodiazepine ("PBD") dimers. PBD dimers bind to the DNA of tumor cells to block cell division, the process by which cancer spreads.

b. Linker: Seattle Genetics claims that SGN-CD33A "employs a novel linker system and proprietary, site-specific conjugation technology (EC-mAb) that allows uniform drug-loading of the cell-killing PBD agent to the anti-CD33 antibody."[3]

c. Antibody: SGN-CD33A antibodies target a receptor called CD33, which is expressed on AML cancer cells.

38. According to Seattle Genetics, this design allows SGN-CD33A "to be stable in the

[3] *See* http://www.seattlegenetics.com/pipeline/vadastuximab-talirine.

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bloodstream and to release its potent DNA binding agent upon internalization into CD-33 expressing cells."[4]

39. Seattle Genetics has worked on PBD dimer payloads since 2008 under an exclusive licensing arrangement with Spirogen. Spirogen is an ADC developer based in the United Kingdom, which was acquired by the AstraZeneca Group in 2013.

40. SGN-CD33A is a successor to SGN-33 and Mylotarg.

41. Mylotarg was an ADC developed by Pfizer. It was approved in 2000 with a "black box" warning label that stated, in relevant part, "Hepatotoxicity, including severe hepatic veno-occlusive disease (VOD), has been reported in association with the use of Mylotarg as a single agent . . . ." After post-marketing surveillance demonstrated that the rate of VOD was even higher than expected, and a post-approval clinical trial showed no clinical benefit, Pfizer voluntarily withdrew Mylotarg from the market in June 2010.

42. SGN-33, a predecessor to SGN-CD33A, was an ADC that targeted CD-33 cells found on leukemic cells. Several years before the Class Period, Seattle Genetics initiated a Phase II trial that enrolled 211 previously untreated AML patients age 60 or older who were either ineligible for or declined intensive chemotherapy. The trial's ultimate endpoint sought to evaluate whether a combination of SGN-33 with a chemotherapy called cytarabine could extend overall survival compared to cytarabine administered with a placebo.

43. On September 13, 2010, Seattle Genetics announced in a press release that it would discontinue the development of SGN-33 because the Phase II trial did not meet its primary endpoint of extending overall survival. On the same day, Defendant Siegall held a conference call to announce the Company's decision to abandon SGN-33, and stated that the "drug failed us."

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[4] *See* http://www.seattlegenetics.com/SGN-CD33A.

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44. Beginning in July 2013, Seattle Genetics began clinical trials of SGN-CD33A. Its initial clinical trial was a Phase I open-label study of SGN-CD33A in combination with hypomethylating agents ("HMA Phase I"). The full name of the HMA Phase I study is: "A Phase 1 trial of SGN-CD33A in Patients With CD33-positive Acute Myeloid Leukemia." HMAs, including decitabine or azacitidine, are considered standard treatment for older patients with AML. The trial also evaluates anti-leukemic activity, pharmacokinetics and overall survival in patients with AML. On the basis of interim data from the HMA Phase I, in May 2016, Seattle Genetics initiated a Phase III, randomized, double-blind, placebo-controlled clinical trial called CASCADE. The CASCADE trial is designed to determine if SGN-CD33A in combination with the HMAs can extend overall survival in older patients with AML compared to patients treated with HMAs alone. In December 2016, the FDA placed the HMA Phase I trial of SGN-CD33A on a partial clinical hold.

45. In December 2014, Seattle Genetics initiated a Phase I study to evaluate SGN-CD33A administered in combination with a chemotherapy regimen known as 7+3 for younger patients with newly diagnosed AML ("7+3 Phase I"). The 7+3 Phase I study is entitled: "A Phase 1b Dose Escalation Study of SGN-CD33A in Combination With Standard-of-care for Patients with Newly Diagnosed Acute Myeloid Leukemia." This open-label clinical trial seeks to determine the maximum tolerated dose and safety profile of SGN-CD33A. It also seeks to evaluate anti-leukemic activity, pharmacokinetics and overall survival. In December 2016, the FDA placed the 7+3 Phase I trial on a partial clinical hold.

46. In November 2015, Seattle Genetics initiated the Stem Cell Phase I/II trial, an open-label clinical study of SGN-CD33A in patients with relapsed or refractory AML. This study was titled: "A Phase 1/2 Study of Vadastuximab Talirine Administered in Sequence With Allogeneic

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Hematopoietic Stem Cell Transplant in Patients With Relapsed or Refractory Acute Myeloid Leukemia (AML)." This trial sought to evaluate SGN-CD33A as a pre-conditioning regimen before the administration of an allogenic stem cell transplant and as a maintenance therapy after a stem cell transplant. On December 27, 2016, Seattle Genetics announced that the FDA placed the Stem Cell Phase I/II trial on full clinical hold. On March 6, 2017, Seattle Genetics announced that it would abandon this trial, citing challenges associated with developing an effective treatment in this setting.

47. Even before the Class Period, Defendants claimed that SGN-CD33A's superior linker and cytotoxic payload differentiated SGN-CD33A from Mylotarg's known deficiencies. For example, on January 11, 2016, Defendant Siegall attended the J. P. Morgan Health Care Conference and made the following claims regarding the difference between SGN-CD33A and Mylotarg:

> **<Q>:** [Question Inaudible] (09:17-09:30)
>
> **<A - Clay B. Siegall>:** Right. The question from [ph] Richard (09:32) was about this new ADC drug conjugate, 33A, why are we more excited than Mylotarg, which was a 33 drug conjugate of the past. Mylotarg had a lot of issues and problems, but it was a phenomenal pioneering molecule.
>
> . . .
>
> And when you think about Mylotarg, the biggest toxicity that with a problem, and it was written about a lot was veno-occlusive disease. And we've treated couple of hundred patients that with 33A without VOD. And so, we –you just don't see the same toxicity that was probably because the drug was falling off very readily, a drug being calicheamicin. And so, I think we have used all our engineering skill to make what I think is one of the best ADC technologies there is. And we put it into this – using this target 33, which we like a lot. And despite all of the problems that Mylotarg had, it still had some really nice subjective responses. It still had some survival data. Not huge, but had some despite its problems in toxicities. And so, I just think that we used the right target with all the new technologies. Is there anything else to add to that? Okay. Yeah.

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Known Hepatotoxicity Associated with SGN-CD33A

1. Confidential Witnesses

48. Prior to the start of the Class Period, Seattle Genetics and the Individual Defendants were well aware that SGN-CD33A posed a high risk of hepatotoxicity. According to Confidential Witness 1 ("CW1"), hepatotoxicity was known, *inter alia*, from prior experience with a predecessor drug using similar components, from internal Safety Data Sheets made available to Seattle Genetics employees, and from a third-party risk assessment.

49. CW1 has seventeen years of experience in the biotechnology industry, and served as the Senior Environmental Health and Safety Engineer at Seattle Genetics from March 2015 to February 2017. As Senior Environmental Health and Safety Engineer at Seattle Genetics, CW1 was responsible for providing information to Seattle Genetics employees about the risks of the environment in which they worked, including the risks of exposure to toxic drug compounds in Seattle Genetics labs, and the stringent handling requirements for those drugs. CW1 primarily dealt with the division at Seattle Genetics that was responsible for synthesizing drugs, including SGN-CD33A. CW1 reported to David Moore ("Moore"), the Associate Director of Facilities, and Tina Bailey, the Senior Manager of Facilities, at Seattle Genetics. Moore reported to Mike Mabrito, the Director of Facilities at Seattle Genetics, and both Moore and Mabrito directly reported to Defendant Simpson.

50. According to CW1, Seattle Genetics collaborated with Spirogen to initiate a clinical trial that enrolled patients on a standalone therapy similar to SGN-CD33A. Early data from animal studies for this predecessor drug indicated high risk of treatment-related hepatotoxicity, and Seattle Genetics ultimately terminated the clinical trial. CW1 explained that the development of SGN-CD33A was based on a similar molecule to the one utilized in the earlier trial, and "almost all of

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the safety correlations [with SGN-CD33A] were based off that Spirogen compound."

51. Because SGN-CD33A and the predecessor that the Company developed in collaboration with Spirogen were similar, the data from the predecessor was used to develop the safety protocols that CW1 was responsible for communicating to Seattle Genetics' various divisions. According to CW1, the animal safety studies for the predecessor drug were initiated to assess the safety risks that might be observed in human clinical trials, not to assess environmental or occupational safety risks.

52. CW1 coordinated with the Company's in-house toxicologist to prepare Safety Data Sheets that listed specific levels of toxicity associated with each organ in the human body. According to CW1, the Safety Data Sheets specifically contained information about how specific levels of toxicity could damage organs, including the liver. These reports were available to the Company's officers, including the Individual Defendants, and any employees authorized to access the reports on the Company's Intranet known as "The Linker." For SGN-CD33A, the Safety Data Sheets indicated a risk of hepatotoxicity.

53. According to CW1, in the middle of 2016, Seattle Genetics procured a third party risk assessment of the toxicity associated with SGN-CD33A, and that assessment concluded that the risks were high. CW1 explained that Lonza, a contract manufacturer that Seattle Genetics used to manufacture the cytotoxic payload and linker components of SGN-CD33A, learned of the third-party risk assessment, and suspended manufacturing these components as a result.

54. CW1 was asked to collaborate with the in-house toxicologist to respond to the third party's risk assessment in an effort to convince Lonza to continue production of SGN-CD33A's components.

55. After CW1 raised concerns about the risks of exposure to SGN-CD33A, CW1 was

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instructed by Mabrito not to discuss the issue with the in-house toxicologist, and the Company threatened to fire CW1 if he violated this directive.

56. CW1 stated that Seattle Genetics' in-house toxicologist initially also expressed concerns about SGN-CD33A's level of toxicity (consistent with the findings of the third-party assessment), but the in-house toxicologist was coerced to moderate his views by Moore and Mabrito. CW1 understood this coercion originated from Defendant Simpson.

57. CW1 approached several senior-level officers in the compliance and human resources divisions within the Company about his concerns with no success. CW1 stated that these concerns were eventually communicated to Seattle Genetics' General Counsel. CW1 attempted to directly reach Defendant Simpson to discuss his concerns, but Jodi Jamieson, Seattle Genetics' Senior Director of Human Resources and Organizational Development, told CW1 that Simpson would not meet with CW1. CW1 also emailed Defendant Siegall's executive assistant to seek a meeting, and copied the executive assistant on various emails with Human Resources. However, Defendant Siegall did not respond to CW1.

58. Confidential Witness 2 ("CW2") was a Senior Scientist at Seattle Genetics from 2010 to July 2017. CW2 worked extensively on the chemistry of SGN-CD33A and helped to synthesize the drug for human trials. CW2 described SGN-CD33A as "the most toxic drug in the history of the world." According to CW2, Seattle Genetics rushed SGN-CD33A to clinical trials without fully understanding the risk of hepatotoxicity associated with the drug.

59. CW2 explained that everyone at Seattle Genetics knew that SGN-CD33A was very similar to Mylotarg. As a result, CW2 and other scientists were not surprised when hepatotoxic events were observed in SGN-CD33A's clinical trials. CW2 explained that Pfizer used lower doses more frequently with Mylotarg in an effort to limit liver toxicity. However, CW2 stated that

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Seattle Genetics was very aggressive, and increased dosage as high as possible without fully understanding the risk of hepatotoxicity.

60. According to CW2, Defendants Siegall, Simpson and Drachman are micromanagers, who "knew the most intimate details [about] Mylotarg." CW2 recalled that he attended company-wide meetings that were also attended by Defendants Siegall, Simpson and Drachman. CW2 stated that, at these company-wide meetings, the Company expressed deep concerns about the hepatotoxicity associated with SGN-CD33A before the press releases about the trials were issued to the public. CW2 asserted that the clinicians knew about the risks of hepatotoxicity associated with SGN-CD33A, and these clinicians ultimately reported to Defendant Siegall.

61. CW2 further stated that Eric Feldman, Seattle Genetics' former Senior Medical Director and Distinguished Clinical Scientist, was aware of the risk of hepatotoxicity associated with SGN-CD33A and SGN-CD33A's similarity to Mylotarg. CW2 explained that Feldman oversaw the SGN-CD33A clinical trials, and directly reported to Defendant Siegall.

62. According to CW2, the stakes with SGN-CD33A were particularly high because SGN-CD33A was Seattle Genetics' most advanced Phase III drug candidate, and its failure would mean that the Company "would have nothing else in the pipeline."

2. Individual Defendants Claimed To Be Familiar with SGN-CD33A's Toxicity

63. In their statements to investors, Individual Defendants spoke at length (albeit falsely) about the safety profile of SGN-CD33A, indicating both that they understood the importance of toxicity to investors and that they understood they were holding themselves out as having knowledge of toxicity.

64. For example, on January 7, 2013, Defendant Siegall attended the J.P. Morgan Healthcare Conference, and claimed to be familiar with the increased potency of the Company's

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newer ADCs, and conceded he was "very familiar and aware" of toxicity problems with Mylotarg, a predecessor to SGN-CD33A:

> But going back to the CD33 product, that one employs our next generation ADC technology. It's a different chemo type. Our chemo type in ADCETRIS affects tubulin so it affects cell division through tubulin. The new product affects DNA directly. It's more potent. It's a couple of hundred times more potent than auristatin which is the component, the drug component of ADCETRIS.
>
> We developed a new linker. We developed an engineered antibody that has two attachment sites so it's a highly specific second generation antibody-drug conjugate that we've developed for CD33. We're very familiar and aware of the data with Mylotarg with CD33, how it did help some patients but had some level of toxicity. The linker for that product was not very stable. It was developed a few decades ago. We think we've developed a product that has a fantastic preclinical package and we're excited to get that into clinical trials this year.

65. On May 21, 2013, Defendant Siegall attended the UBS Global Healthcare Conference. Again he emphasized the potency of the new dimer, or warhead, used in SGN-CD33A, describing it as "250 times more potent" than the dimer used in the Company's approved drug:

> Now, going back and talking about CD33 drug conjugate for a moment. This uses a new technology that we've never put in humans. It uses a PBD drug, pyrrolobenzodiazepine dimer. It's about 250 times more potent than auristatin, which is the drug we use in ADCETRIS.
>
> And we developed a new antibody engineering technology. We call it EC, or engineered cysteine, mAbs. We developed a new linker. So, it's completely brand new technology. The pre-clinical package in AML cells is extraordinary. I've really like it, but we haven't treated a patient yet. That will start this year. Within the next few months, we'll be treating patients, and I really hope that we can make an impact on AML.

66. On June 12, 2013, Defendant Siegall attended the Goldman Sachs Healthcare Conference, and again claimed to be knowledgeable about the preclinical package for SGN-CD33A:

> I'm excited about a lot on our pipeline, but if I have to bring up one we have a CD33 drug conjugate that we filed for approval two months ago, filed for IND –

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> excuse me two months ago it starts in clinical trial and I'm very excited about it because our preclinical data targeting AML patients is extraordinary. Our preclinical package, I really am excited about, and I could compare it and say it has a preclinical package as exciting as ADCETRIS had, but not with preclinical and AML is a wicked disease, it is one of the worst cancers, I would say, arguably the worst hematologic or blood cancer.
>
> I would put it akin to pancreatic cancer a solid tumor for how many patients die quickly from it, and I, in my career, I want to make a difference in those patients life. And so I'm really excited with the preclinical package and going after CD33 as the target for AML. It's literally on every AML patient. So it is the right target and we built the right drug conjugate and we're putting our money, where our mouth is.

The preclinical package for SGN-CD33A with which Siegall claimed familiarity included animal studies demonstrating severe toxicity, *see* ¶¶ 50-51.

67. On July 31, 2013, Seattle Genetics announced financial results for Q2 2013, and held a conference call, in which Defendant Siegall again confirmed his knowledge of SGN-CD33A preclinical studies:

> Now, the second part of your question is on AML and our SGN-CD33A drug conjugate. As I've said before, the pre-clinical data set for this product candidate is amongst the best I've ever seen in my career. I'm really excited about it. But I'm also tempered by it's just pre-clinical.

68. On November 5, 2013, Seattle Genetics announced financial results for Q3 2013, and held a conference call, in which Defendant Siegall acknowledged the failure of SGN-33, the failed predecessor of SGN-CD33A, but assured investors that the Company "had a learned a ton" from the predecessor's failure, including "what we needed and what kind of potency we needed" to assure SGN-CD33A's success:

> As you know, we've been interested in AML for many years. We developed a product that didn't work in AML, SGN-33, the initial antibody. That was a naked antibody, and we really worked hard at some very good studies, but it didn't pan out for the patients. But we learned a ton, and we learned really what we needed and

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> what kind of potency we needed. And sometimes it's okay to fail if you learn a lot from it and grow from it.
>
> And I think that our experience with targeting AML really gave us a lot of insight into the disease and what was needed. And we worked for many years to develop the CD33A drug conjugate, and it's got all of this different new technology in it. It's got a new linker, a new payload. It's got a new engineered antibody system we call EC-mAbs, or engineered cystine mAbs. So I think you should expect data in 2014 on that.

69. On November 20, 2013, Defendant Siegall attended the Jefferies Healthcare Conference. At this event, Defendant Siegall again claimed to understand the preclinical studies for SGN-CD33A, the need to achieve better safety and efficacy than Mylotarg, and the potency of SGN-CD33A:

> We also are using a new payload that's different than what we have in ADCETRIS. We are using a PBD, a pyrrolobenzodiazepine dimer. It's about 200 to 300 times more potent than monomethyl auristatin E or MMAE, and it's not sensitive to multi-drug resistance. And we developed this drug and put it into preclinical models. And we compared it to what you see on this slide, GO is gemtuzumab ozogamicin, or Mylotarg, which was on the market targeted to CD33.
>
> … And what we've shown is that you can get substantially better activity at low well-tolerated doses because we're using a newly engineered antibody, a stable linker, which was one of the issues with Mylotarg, a highly potent synthetic drug, not a natural product.
>
> So we've really put all of the new innovations from Seattle Genetics' research efforts for over six years into this molecule, and the preclinical package is one of the best I've ever seen. And we are in clinical trials and doing dose escalation, and looking forward to presenting data from this drug in 2014.

70. On July 31, 2014, Seattle Genetics announced Q2 2014 financial results and held a conference call, in which Defendant Drachman affirmatively stated that a primary goal of the preliminary trials for SGN-CD33A was to monitor the level of toxicity:

> **<Q - Thomas A. Wei>**: … And then my question on AML is just an understanding of what we should expect from that data. The protocol's a mix of naïve patients

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who are ineligible for chemotherapy and then relapsed patients who achieve a complete response in the first-line setting. And I'm just trying to understand what is the right benchmark for judging efficacy in each of those populations. Thanks

<A - Clay B. Siegall>:… Concerning AML and expectations, that's a hard question early on in Phase 1 when you're really learning about the safety profile and you're learning about dose and schedule and the different patient types. So I don't want to give you too much of specifics of what you exactly will be looking for in here and any specific benchmarks.

Keep in mind, important benchmarks with AML are really survival and that's something that you get in larger studies and randomized studies and certainly that's what the FDA expects for approval for AML-type drugs. And Jonathan, would you like to make any remarks on AML?

<A - Jonathan Drachman>: ***I just agree with what Clay said that we're learning – we're going to be learning a lot about a new chemo type in this trial, the PBD dimer, and learning about tolerability, activity, and toxicities.*** As far as the AML goes, it's a terrible disease. There really haven't been any successful advances for decades. And in the two populations that we're looking at, nothing really works. You've got older patients who can't get intensive therapy with curative intent at all, so they get palliative therapies at best, and then you've got patients who have failed their best chance of cure in frontline and relapsed. So it's hard to say what the benchmarks are in a Phase 1 population, and I think you'll have to judge from the data when you see it.

71. On October 29, 2015, Seattle Genetics announced financial results for Q3 2015, and held a conference call, in which Defendant Drachman acknowledged that he understood the importance of avoiding toxicity in SGN-CD33A:

<Q - Adnan S. Butt>: Good. Clay, if I can just get a follow-up here. Some other – a number of other agents are being combined with HMAs and AML. So when we look at this data to understand it best, do we look at the kinds of patients that Seattle Genetics is targeting? Do we look at the response rate? How do we gauge, what is meaningful in the abstracts and at ASH?

<A - Clay B. Siegall>: Yeah. Well, I think that as far as patients go, Adnan, we're looking at frontline patients. So these are newly diagnosed and are unfit for therapy. And Jonathan can give a little bit more color on the specific patients there. We will provide as much data as we can, in our abstract and in our presentation. But please

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note, that these patients on this arm, if you will, were enrolled during 2015. So, we are following these as close as possible; we're encouraged by the data we see. And Jonathan, would you like to add anything more?

<A - Jonathan Drachman>: No. I think that Clay really covered most of it. And recall that the type of patients who are generally treated with frontline HMA monotherapy are the ones who tend to be older, who have worse cytogenetics, who don't have curable disease. ***And so, without speaking to exactly the population that we treated and will be in our abstract, we're treating a frail, older population, where the goal is to provide a meaningful improvement in their outcomes without adding a lot of toxicity. And that's one of the things that's really exciting about a targeted agent that can deliver a leukemic cell-killing payload without causing a lot of toxicity.***

72. On April 28, 2016, Seattle Genetics announced the financial results for Q1 2016, and held a conference call. At this event, Defendants Siegall and Drachman again conceded the importance of distinguishing SGN-CD33A's safety profile from Mylotarg:

<Q - Boris Peaker>: Great. My first question is on 33A. As you try to bring on clinical sites on board and talk to physicians and trial participants, I'm wondering, do they compare it to Mylotarg? Do they see it as maybe like Mylotarg version 2.0? And do you have data to show them in terms of consistency of manufacturing, or how it maybe is different from Mylotarg so that would not stop them in any way?

<A - Clay B. Siegall>: Yeah. When we started 33A, I think that some docs said, well, explain to us what this drug is – and these AML docs are all very aware of Mylotarg. And in fact, most of them were not happy that Mylotarg was taken off the market because they viewed Mylotarg as being an effective drug for a lot of patients. And so they were very disappointed that it wasn't on the market.

And so a lot of them asked questions, and then as we got into this, we have a highly engineered antibody, we have two direct attachment sites, we have a novel chemo type that's outside of MDR, which calicheamicin wasn't. We have a stable linker. And maybe that's the most critical of all the pieces, that the drug and the antibody were staying together in the bloodstream until it found the tumor, where with Mylotarg it clearly had a very short half-life and it was not outside of MDR and there was a lot of side effects from Mylotarg.

So, when you look at the kind of data we have and the side-effect profile we have, it's like a different drug completely. The similarity rests with the targeting of CD33,

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> but outside of that, it's a completely different drug. And I think that we used to hear some comments about Mylotarg when we started the trial, and I don't think we hear them anymore.
>
> Now, we hear more about the drug and the data and its activity. And it's something that's – there's a lot of excitement in the oncology and in the hem/onc community of the leukemia docs treating this devastating disease. So accrual's been great with 33A. I mean, we have docs that are really working hard to try to be on our trial. So we have a lot of excitement there. Jonathan, do you want to add anything?
>
> **<A - Jonathan Drachman>**: I think Clay summarized it very well. This is a different drug, and I think the main way that people think about Mylotarg is really validating CD33 as an important target for AML.

3. Individual Defendants Knew of Hepatotoxic Events as They Occurred

73. In addition to knowing the undisclosed *risks* of hepatotoxicity, Defendants also learned of the six specific hepatotoxic events at issue here, including four deaths, as they occurred in the open-label Phase I trials of SGN-CD33A. Seattle Genetics, as the sponsor of these trials, was required to report each of those events to the FDA within seven (7) days. While the exact timing of each hepatotoxic event is not known, Plaintiffs are informed and believe that they occurred at least several weeks, if not months, prior to the end of the Class Period.

74. Plaintiffs base this information and belief on the following facts: (a) it is highly improbable that six separate hepatotoxic events in trials that had been ongoing since 2013-2015 suddenly occurred at a single moment in time in late 2016. An even distribution of those events during the time of the clinical trials would suggest that multiple adverse hepatotoxic events had already occurred prior to the Class Period; (b) even if clustered towards the end of the Class Period, a pattern of delay in reporting adverse events for SGN-CD33A demonstrate that the hepatotoxic events likely occurred before most if not all of the alleged Class Period statements, especially the December 2016 press releases alleged herein, *see* ¶ 77; and (c) in addition to the observed pattern of delay, the mechanics of reporting adverse events and clinical holds demonstrates that several

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weeks would likely pass between the events and the December 27, 2016 clinical holds.

75. A small subset of the serious adverse events related to SGN-CD33A that is publicly available demonstrates a pattern of delayed reporting.

76. The FDA Adverse Event Reporting System (FAERS) is a publicly-available data-base that summarizes certain adverse event reports that have been submitted to the FDA.

77. For SGN-CD33A, FAERS provides information about 24 serious adverse events, including 14 deaths, related to the use of SGN-CD33A in combination with the HMA agent known as azacitidine. This HMA agent was used in several clinical trials for SGN-CD33A. The following table shows that the average delay between the date of a serious adverse event and the date that event was reported to the FDA exceeded 41 days, and the median delay was 34 days:

Event Date of SAE	SAE Date Received by the FDA	Number of Days Between Event Date and Date Received by the FDA	Patient Outcomes
06/24/2017	07/24/2017	30	Hospitalized
4/6/2017	6/2/2017	57	Died; Hospitalized
4/18/2017	6/13/2017	56	Died; Hospitalized
4/21/2017	5/31/2017	40	Died; Hospitalized; Other Outcomes
7/16/2017	8/18/2017	23	Died; Hospitalized
7/14/2017	8/9/2017	26	Hospitalized
6/6/2017	7/18/2017	42	Died; Hospitalized
5/15/2017	7/19/2017	65	Hospitalized
6/30/2017	8/9/2017	40	Died; Hospitalized
5/12/2017	7/13/2017	62	Hospitalized
3/27/2017	5/18/2017	52	Hospitalized; Life Threatening; Other Outcomes
4/4/2017	4/27/2017	23	Hospitalized

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12/29/2016	3/29/2017	90	Hospitalized
6/22/2017	7/10/2017	18	Hospitalized; Life Threatening
1/21/2017	2/28/2017	38	Died; Hospitalized
4/13/2017	5/9/2017	26	Died; Hospitalized; Life Threatening
6/2/2017	6/21/2017	19	Died; Hospitalized; Life Threatening
2/14/2017	5/23/2017	98	Died; Hospitalized
5/6/2017	5/23/2017	17	Died; Hospitalized; Life Threatening
2/5/2017	2/28/2017	23	Disabled; Hospital-ized
Unknown	5/3/2017	Unknown	Died; Hospitalized
11/30/2016	3/21/2017	111	Hospitalized
Unknown	12/6/2016	Unknown	Died; Hospitalized
9/17/2016	10/17/2016	30	Died; Hospitalized

78. Accordingly, even if the six serious hepatotoxic events were not evenly distributed throughout the SGN-CD33A clinical trials, but instead were weighted towards the final months of 2016, the pattern and practice of delayed reporting suggests that some or all of these events almost certainly preceded the alleged Class Period misrepresentations.

79. Even if it did not delay reporting, Seattle Genetics was permitted a week after each potentially fatal event before it was required to report the event to the FDA, after which the FDA would require time to process and review the adverse event reports. Once the FDA decided to consider initiating clinical holds, regulations specified that it should generally attempt to resolve the issue with the sponsor before initiating the hold, all of which suggests that the adverse events here were part of a chain of events that began prior to December 2016. *See* 21 U.S.C. § 312.32; 21 C.F.R. § 312.42(c); *see also* ¶¶ 30-31.

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4. Individual Defendants Had Actual Knowledge of Hepatotoxicity Problems From Their Experience with ADCETRIS

80. When the FDA approves a new drug, it also reviews and negotiates with sponsors a specific label containing prescribing information that must accompany all marketing of the drug. The label of a drug is significant because it can influence prescriptions for the drug. Accordingly, it is considered by investors and analysts, and is core information for executives overseeing drug marketing. For Seattle Genetics, the label negotiated for its sole approved drug, ADCETRIS, emphasized hepatotoxic risk. It states that "[s]erious cases of hepatotoxicity, including fatal outcomes, have occurred in patients receiving ADCETRIS. Cases were consistent with hepatocellular injury, including elevations of transaminases and/or bilirubin. Cases have occurred after the first dose of ADCETRIS or after ADCETRIS rechallenge. Preexisting liver disease, elevated baseline liver enzymes, and concomitant medications may also increase the risk. Monitor liver enzymes and bilirubin. Patients experiencing new, worsening, or recurrent hepatotoxicity may require a delay, change in dose, or discontinuation of ADCETRIS." Defendants held themselves out to investors as having familiarity with the ADCETRIS label. For example, Seattle Genetics' Form 10-K dated February 29, 2012, which was signed by Defendants Siegall and Simpson, discusses ADCETRIS labeling in several places, and shortly before ADCETRIS was approved, Defendant Siegall confirmed to an analyst, during a Q2 2011 earnings conference call, that the Company had extensive discussions with the FDA on a variety of topics, including ADCETRIS labelling.

DEFENDANTS' MATERIALLY FALSE AND MISLEADING STATEMENTS AND OMISSIONS DURING THE CLASS PERIOD

81. The Class Period begins on October 27, 2016. On a conference call that day held in connection with the Company's earnings report for the third quarter of 2016, Defendant Drachman made the following misrepresentations in response to an analyst question about the results of

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the 7+3 Phase I study:

> **<Q - Cory W. Kasimov>**: Hey, good afternoon, guys. Thanks for taking the questions. I guess, first, to follow up on the SGN-CD33A program. With the data that we're going to see at ASH in combination with 7+3, are you able to say roughly how many patients or how much follow-up we might see in San Diego?
>
> **<A - Clay B. Siegall>**: I'll turn that over to Jonathan and see what Jonathan is willing to tell you.
>
> **<A - Jonathan Drachman>**: All right. So, Cory, thanks for the question. We can't disclose exactly the details of the number of patients or the follow up. The study has been going on for a while, not a huge amount of time. And just as a reminder to people about the frontline younger patients and what the goal is. ***This is really an area where more than half the patients are treated aggressively with 7+3 or a similar regimen with an intent to try to cure those patients. There're pretty high CR [complete remission] rates, and there is a substantial number of patients who are cured with 7+3 consolidation with or without allogeneic transplant in remission.*** So, it is a complicated space. It's really like trying to redefine frontline therapy with curative intent. ***So it's something that we're looking at really closely. We're excited about our interim data.*** And when we present it, I think it'll be a good time to evaluate whether we feel like we're making a difference at that time and then what our next steps will be.

82. The statements identified in paragraph 81 were materially false and misleading when made because they omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were experiencing serious adverse hepatotoxic events.

83. On the October 27, 2016 conference call, Defendant Siegall made the following materially false and misleading statements in response to a question from an analyst concerning the difference between SGN-CD33A and AML therapies under development by Seattle Genetics' competitors:

> **<Q - Tazeen Ahmad>**: Hi. Good afternoon. Thanks for taking my question. One

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> on SGN-CD33A, if I might, just to follow up on some of the conversations we've been having so far on the call. Obviously, this is going to be something that we're all going to be looking for at the ASH meeting, but in terms of how you're thinking of its profile versus other drugs that are in development, there are few companies that are trying to go for similar populations in AML, whether it be AbbVie, Otsuka, Boehringer or a number of other companies. What should we really be looking for? Should we be looking for differences in efficacy or safety at this early stage?
>
> **<A - Clay B. Siegall>**: . . So, there are few [competing drug candidates], but as far as a drug targeted with an antibody and delivering cytotoxic like our antibody-drug conjugate, ***we're very happy with our positioning in this field and think that this could make a big difference for patients. And because SGN-CD33A is expressed on all these tumors that with AML, basically all of them could be very user-friendly from a combination standpoint, much like ADCETRIS is user-friendly from a combination standpoint in Hodgkin lymphoma.*** Because CD30 is expressed on all Hodgkin lymphoma, basically, and we could use it in combination with other types of therapies, whether they be old-school therapies, like cytotoxics or newer therapies like some of the checkpoint inhibitors. So, we think that ADCs make extraordinary regimen partners, if you will, with other drugs. And we're going to continue doing that.

84. The statements identified in paragraph 83 were materially false and misleading when made because they omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were experiencing serious adverse hepatotoxic events.

85. On November 8, 2016, Defendant Siegall attended the Credit Suisse Health Care Conference and made the following materially false and misleading statements:

> . . . We're taking this very active single agent drug that we presented more than a year ago and combining it with hypomethylating agents, which are used to treat older AML patients. And what you could see on this slide is a 71% CR/CRi rate, which is way higher than you would see with HMAs alone.
>
> And we're excited with the data. The median OS is interim and is moving around, but what's really important is that we have a low 30 and 60-day mortality rate,

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which we see in these patients and the data that we have presented really support going forward into our phase 3 CASCADE trial.

. . .

And so we know that there's a good safety profile there and we'll be presenting data at the ASH conference to show the detailed safety and efficacy of what we've come up with so far.

86. The statements identified in paragraph 85 were materially false and misleading when made because there was not a "good safety profile" for SGN-CD33A, and because the statements omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were experiencing serious adverse hepatotoxic events.

87. On December 3, 2016, Seattle Genetics issued a press release announcing partial results from the 7+3 Phase I study, which included the following statements:

Seattle Genetics Presents Phase 1b Data from Vadastuximab Talirine (SGNCD33A; 33A) in Combination with Standard of Care in Frontline Acute Myeloid Leukemia at ASH Annual Meeting

-Clinical Data Featured in Press Program and Oral Presentation Indicate 33A is Well Tolerated in Combination with 7+3 Induction Therapy in Younger Newly Diagnosed AML Patients

-Antileukemic Activity Data Show Remission Rate of 76 Percent, with 78 Percent of Those Remissions Negative for Minimal Residual Disease-

. . .

"Our clinical trial data reported at ASH demonstrate that adding vadastuximab talirine, also known as 33A, to standard of care treatment results in a rapid, high rate of remissions in frontline, younger AML patients with poor prognosis. Notably, seventy-eight percent of patients who achieved remissions in this trial tested negative for minimal residual disease, which means no cancer could be detected with a sensitive test," said Jonathan Drachman, M.D., Chief Medical Officer and Executive

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Vice President, Research and Development at Seattle Genetics. ***"In this trial, 33A in combination with 7+3 was well-tolerated, with a low early mortality rate***. Based on these promising, early data, we plan to initiate a randomized phase 2 clinical trial in 2017 in younger newly diagnosed AML patients to further evaluate the potential benefit of adding 33A to standard of care."

"People with acute myeloid leukemia die of infections or bleeding within weeks or a few months of diagnosis without effective, aggressive chemotherapy. Even with current treatment regimens, fewer than 50% of younger adults are successfully treated. ***The phase 1 results of 33A in combination with standard of care show a high rate of remissions in younger newly diagnosed AML patients without significantly adding to the toxicity of the treatment.***

. . .

Data were reported from 42 newly diagnosed AML patients with a median age of 46 years and intermediate or adverse cytogenetic risk of 50 percent and 36 percent, respectively. Seventeen percent of patients had secondary AML. Key findings include:

. . .

- ***The most common Grade 3 or 4 treatment-emergent adverse events occurring in 20 percent or more of patients were febrile neutropenia, thrombocytopenia, anemia and neutropenia. No non-hematologic treatment-emergent adverse events of Grade 3 or higher were reported in 15 percent or more of patients. No veno-occlusive disease/sinusoidal obstruction syndrome or significant hepatotoxicity was observed on treatment.***

- ***The most common Grade 1 and 2 treatment-emergent adverse events occurring in 20 percent or more of patients were nausea, diarrhea, constipation, hypokalemia and decreased appetite. No infusion-related reactions occurred.***

. . .

88. The statements identified in paragraph 87 were materially false and misleading when made because they omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials

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were experiencing serious adverse hepatotoxic events, including veno-occlusive disease, even if the observations did not occur in this particular trial.

89. On December 5, 2016, Seattle Genetics issued a press release announcing partial results from the HMA Phase I study, which included the following statements:

> *– Both Combination and Monotherapy Data Show 33A is Well-Tolerated with Rapid, High Remission Rates for AML Patients in Multiple Phase 1 Trials; Data Highlighted in Three Oral Presentations –*
>
> . . .
>
> ***"We are pleased with the growing body of data demonstrating that vadastuximab talirine, also known as 33A, has a promising overall tolerability and activity profile in clinical trials for patients with AML," said Jonathan Drachman, M.D., Chief Medical Officer and Executive Vice President, Research and Development at Seattle Genetics.***
>
> . . .
>
> Data were reported from 53 frontline AML patients with a median age of 75 years and predominantly intermediate or adverse cytogenetic risk who had declined intensive therapy. Regarding additional poor-prognosis indicators, 42 percent of patients had evidence of underlying myelodysplasia, 11 percent had FLT3-mutated disease and 43 percent had secondary AML, which is AML that arises from prior chemotherapy, a pre-existing MDS or myeloproliferative disease. Key findings include:
>
> . . .
>
> - With a median follow-up of 14.7 months, median overall survival for all patients was 11.3 months and 28 percent of patients remained alive and on study as of last follow-up. The 30- and 60-day mortality rates were two and eight percent, with no treatment-related deaths occurring during that time.
>
> . . .
>
> - ***The most common Grade 3 or 4 treatment-emergent adverse events occurring in 20 percent or more of patients were thrombocytopenia, febrile neutropenia, anemia and neutropenia.***

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- ***The most common Grade 1 and 2 treatment-emergent adverse events occurring in 20 percent or more of patients were fatigue, nausea, constipation, peripheral edema and decreased appetite.***

Vadastuximab Talirine Monotherapy in Older Patients with Treatment Naive CD33-Positive Acute Myeloid Leukemia (Abstract #590, oral presentation on Monday, December 5, 2016 at 7:15 a.m. PT)

Interim results from 93 patients in the ongoing phase 1 study evaluating 33A monotherapy in AML patients were previously presented at the 2015 ASH Annual Meeting. New results describing the safety and activity of the recommended 33A monotherapy dose of 40 micrograms per kilogram (mcg/kg) in an expansion cohort of treatment-naïve older AML patients were presented by Dr. Anjali Advani, Cleveland Clinic.

Data were reported from 27 treatment-naïve older AML patients with a median age of 74 years and intermediate or adverse cytogenetic risk of 70 percent and 26 percent, respectively. Regarding additional poor-prognosis indicators, 48 percent of patients had evidence of underlying myelodysplasia and 22 percent had FLT3 mutated disease. Key findings include:

. . .

- ***The most common Grade 3 or higher treatment-emergent adverse events occurring in 20 percent or more of patients were thrombocytopenia, febrile neutropenia and anemia.***
- ***The most common Grade 1 and 2 treatment-emergent adverse events occurring in 20 percent or more of patients were peripheral edema, decreased appetite, fatigue, diarrhea and dizziness.***

90. The statements identified in paragraph 89 were materially false and misleading when made because they omitted the following information necessary to make the statements not misleading under the circumstances under which they were made: (a) that SGN-CD33A had high, known risks of liver toxicity; and (b) as a result, patients exposed to SGN-CD33A in clinical trials were experiencing serious adverse hepatotoxic events.

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The Truth Emerges

91. On December 27, 2016, Seattle Genetics issued a press release, which was also filed with the SEC as an attachment to a Current Report on Form 8-K, disclosing that six patients in SGN-CD33A trials experienced hepatotoxic events, including "several cases of veno-occlusive disease" and four deaths, and that as a result "several Phase I trials" were placed on clinical hold by the FDA:

> **Seattle Genetics Announces Clinical Hold on Several Phase 1 Trials of Vadastuximab Talirine (SGN-CD33A)**
>
> -Enrollment Continues on Phase 3 CASCADE Trial in Acute Myeloid Leukemia and Phase 1/2 Trial in Myelodysplastic Syndrome-
>
> BOTHELL, Wash.--(BUSINESS WIRE)--Dec. 27, 2016-- Seattle Genetics, Inc. (Nasdaq:SGEN), a global biotechnology company, today announced that ***it has received notice from the U.S. Food and Drug Administration (FDA) that a clinical hold or partial clinical hold has been placed on several early stage trials of vadastuximab talirine (SGN-CD33A) in acute myeloid leukemia (AML). The clinical holds were initiated to evaluate the potential risk of hepatotoxicity in patients who were treated with SGN-CD33A and received allogeneic stem cell transplant either before or after treatment. Six patients have been identified with hepatotoxicity, including several cases of veno-occlusive disease, with four fatal events.*** Overall, more than 300 patients have been treated with SGN-CD33A in clinical trials across multiple treatment settings. Seattle Genetics is working diligently with the FDA to determine whether there is any association between hepatotoxicity and treatment with SGN-CD33A, to promptly identify appropriate protocol amendments for patient safety and to enable continuation of these trials.
>
> The phase 1/2 trial of SGN-CD33A monotherapy in pre- and post-allogeneic transplant AML patients has been placed on full clinical hold. Two phase 1 trials have been placed on partial clinical hold (no new enrollment, existing patients may continue treatment with re-consent). These studies are SGN-CD33A monotherapy, including a subset of older AML patients in combination with hypomethylating agents, and SGN-CD33A combination treatment with 7+3 chemotherapy in newly diagnosed younger AML patients. No new studies will be initiated until the clinical holds are lifted.

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> Seattle Genetics' other ongoing trials of SGN-CD33A, including the phase 3 CASCADE trial in older AML patients and phase 1/2 trial in myelodysplastic syndrome, are proceeding with enrollment.

92. On this news, Seattle Genetics' stock price declined by $9.50 per share, or by over 15%, to close at $52.36 on December 27, 2016.

93. Analysts expressed surprise. In particular, Kennen MacKay, a research analyst at Credit Suisse wrote that:

> This morning, SGEN announced several clinical holds imposed by the FDA upon SGN-CD33A ph1 trials driven by cases of [HVOD]. This comes as a surprise to us given: 1) SGN-CD33A was designed to address the [ADC] linker technology pitfalls of its predecessor Mylotarg, thought to be the cause of Mylotarg-associated HVOD, and 2) prior to this report no HVOD concerns had been observed in early stage SGN-CD33A testing. Recall that it was a greater number of HVOD-related deaths which negatively skewed Mylotarg's risk/reward profile and drove Pfizer to voluntarily withdraw the product from the market after having received accelerated approval. Furthermore, while the incidence of Mylotarg's HVOD has been commonly attributed to premature cleavage of the cytotoxic payload, concern continues to exist surrounding potential CD33 target-mediated hepatotoxicity. Given these dynamics, we lower our PoS for SGN-CD33A in frontline older/unfit AML to 30% (from 70% previously) and to 15% in frontline younger/fit AML (from 30% previously), resulting in our ($10) reduction to $60 (from $70 previously), and remain Neutral-rated.

McKay also noted that Defendant Siegall, in a private conversation, declined to specify in which of the Phase I trials the deaths occurred.

Post-Class Period Events

94. On March 6, 2017, Seattle Genetics issued a press release and filed the same with the SEC as an attachment to a Current Report on Form 8-K, announcing that the Company had decided to abandon the Stem Cell Phase I/II trial of SGN-CD33A.

95. The Company also announced that it had implemented a series of risk mitigation measures with respect to hepatotoxicity for all other trials of SGN-CD33A, including modifying

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eligibility standards to exclude patients with liver cirrhosis, and constituting an adjudication committee to verify incidences of HVOD and potentially terminating the treatment if future incidences of HVOD are found. With these measures, the Company indicated that the FDA had agreed to allow it to resume enrollment in the HMA Phase I and 7+3 Phase I trials.

96. On June 19, 2017, Seattle Genetics announced in a press release that the Company would discontinue the Phase III CASCADE trial of SGN-CD33A after reviewing data from the trial and in consultation with an Independent Data Monitoring Committee. According to the press release, the data showed a higher rate of deaths, including unspecified "fatal infections," in the SGN-CD33A experimental arm versus the control arm of the trial. The Company did not explain what was the cause of these alleged "fatal infections."

97. In the June 19, 2017 press release, the Company also announced its decision to suspend patient enrollment and treatment of all SGN-CD33A trials.

98. On June 22, 2017, Seattle Genetics filed a Current Report on Form 8-K, in which the Company disclosed that following the discontinuation of the Phase III CASCADE trial and suspension of all clinical trials of SGN-CD33A, the FDA had placed a hold on the Investigational New Drug Application ("IND") for SGN-CD33A, and that no clinical trials may commence under the IND until the FDA chooses to lift the clinical hold.

99. To this day, all clinical trials of SGN-CD33A remain suspended, and the Company has not provided any substantive details regarding the exact timing of the hepatotoxic events described herein.

CLASS-ACTION ALLEGATIONS

100. Lead Plaintiff brings this action as a class action pursuant to Federal Rule of Civil Procedure 23(a) and (b)(3) on behalf of all persons or entities that purchased or otherwise acquired

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Seattle Genetics' common stock between October 27, 2016 and December 27, 2016, both dates inclusive, seeking to pursue remedies under §§10(b) and 20(a) of the Exchange Act. Excluded are Defendants herein, the officers and directors of the Company, at all relevant times, members of their immediate families and their legal representatives, heirs, successors or assigns and any entity in which Defendants have or had a controlling interest.

101. Class members are so numerous that joinder of all members is impracticable. Throughout the Class Period, Seattle Genetics' common stock was actively traded on the NASDAQ Global Select Market. Because the overwhelming majority of owners hold shares in street name, Lead Plaintiff believes that there are hundreds or thousands of members in the proposed Class. Potential Class members may be identified from records maintained by Seattle Genetics, its transfer agents, and brokers and banks that hold shares beneficially for investors in street name, and may be notified of the pendency of this action by mail, using the form of notice similar to that customarily used in securities class actions.

102. Lead Plaintiff's claims are typical of the claims of those of the Class, as all Class members were similarly affected by Defendants' wrongful conduct in violation of federal law complained of herein.

103. Lead Plaintiff will fairly and adequately protect the interests of the members of the Class and has retained counsel competent and experienced in class action and securities litigation.

104. Common questions of law and fact exist as to all Class members and predominate over any questions solely affecting individual Class members. Among the questions of law and fact common to the Class are:

a. whether Defendants Siegall, Simpson and Drachman are control persons of Seattle Genetics for purposes of the Exchange Act;

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b. whether Seattle Genetics and the Individual Defendants failed to disclose material information regarding SGN-CD33A therapy and its known risk of hepatotoxicity;

c. whether Seattle Genetics and the Individual Defendants made misrepresentations or omissions with scienter;

d. whether the federal securities laws were violated by Defendants' acts as alleged herein;

e. whether the prices of Seattle Genetics' securities during the Class Period were artificially inflated because of the Defendants' conduct complained of herein; and

f. whether the Class has sustained damages as a result of the disclosures alleged herein with respect to their Exchange Act claims and, if so, what is the proper measure of damages.

105. A class action is superior to all other available methods for the fair and efficient adjudication of this controversy since joinder of all members is impracticable. Furthermore, as the damages suffered by individual Class members may be relatively small, the expense and burden of individual litigation make it impossible for Class members to individually redress the wrongs done to them. There will be no difficulty in the management of this action as a class action.

106. With respect to the Exchange Act claims, Lead Plaintiff will rely, in part, upon the presumption of reliance established by the fraud-on-the-market doctrine in that:

a. Defendants made public misrepresentations or failed to disclose material facts during the Class Period;

b. the omissions and misrepresentations were material;

c. Seattle Genetics' securities are traded in efficient markets;

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d. the Company's shares were liquid and traded with moderate to heavy volume during the Class Period;

e. the Company traded on the NASDAQ, and was covered by multiple analysts;

f. the misrepresentations and omissions alleged would tend to induce a reasonable investor to misjudge the value of the Company's securities; and

g. Lead Plaintiff and the Class members purchased and/or otherwise acquired Seattle Genetics' common stock between the time the Defendants failed to disclose or misrepresented material facts and the time the true facts were disclosed, without knowledge of the omitted or misrepresented facts.

107. Based upon the foregoing, Lead Plaintiff and other Class members are entitled to a presumption of reliance upon the integrity of the market.

108. Alternatively, Lead Plaintiff and the Class members are entitled to the presumption of reliance established by the Supreme Court in *Affiliated Ute Citizens of the State of Utah v. United States*, 406 U.S. 128, 92 S. Ct. 2430 (1972), as Defendants omitted material information in violation of a duty to disclose such information, as detailed above.

COUNT I

Violation of § 10(b) of the Exchange Act and Rule 10b-5

(against all Defendants)

109. Plaintiffs repeat and reallege the allegations contained in Paragraphs 1 to 108 above as if fully set forth herein.

110. This Count is asserted against Seattle Genetics and each of the Individual Defendants for violations of Section 10(b) of the Exchange Act, 15 U.S.C. § 78j(b), and Rule 10b-5 promulgated thereunder by the SEC.

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111. During the Class Period, Defendants engaged in a plan, scheme, conspiracy and course of conduct, pursuant to which they knowingly or recklessly engaged in acts, transactions, practices and courses of business which operated as a fraud and deceit upon the Lead Plaintiff and the other members of the Class; made various untrue statements of material facts and omitted to state material facts necessary in order to make the statements made, in light of the circumstances under which they were made, not misleading; and employed devices, schemes and artifices to defraud in connection with the purchase and sale of securities. Such scheme was intended to, and, throughout the Class Period, did: (i) deceive the investing public, including Lead Plaintiff and other Class members, as alleged herein; (ii) artificially inflate and maintain the market price of Seattle Genetics' securities; and (iii) cause Lead Plaintiff and other members of the Class to purchase or otherwise acquire Seattle Genetics' securities and options at artificially inflated prices.

112. Specifically, Seattle Genetics and Defendants Siegall, Simpson and Drachman made material misrepresentations and omissions as particularized in paragraphs 81 to 90.

113. Defendants Siegall, Simpson and Drachman had actual knowledge of the materially false and misleading statements and material omissions alleged herein and intended thereby to deceive Lead Plaintiff and the other members of the Class, or, in the alternative, acted with reckless disregard for the truth in that they failed or refused to ascertain and disclose such facts as would reveal the materially false and misleading nature of the statements made, although such facts were readily available to Seattle Genetics and Defendants Siegall, Simpson and Drachman. In addition to the facts alleged herein demonstrating a strong inference of scienter, certain information showing that Defendants Siegall, Simpson and Drachman acted knowingly or with reckless disregard for the truth is peculiarly within these Individual Defendants' knowledge and control. As the senior managers of Seattle Genetics, these Individual Defendants had knowledge of the details of

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Seattle Genetics' internal affairs, SGN-CD33A and its known risk of hepatotoxicity.

114. As officers and/or directors of a publicly-held company, Defendants Siegall, Simpson and Drachman had a duty to disseminate timely, accurate, full and truthful information regarding Seattle Genetics' business, operations, and financial controls. As a result of the dissemination of the aforementioned false and misleading reports and filings, the market price of Seattle Genetics' securities was artificially inflated throughout the Class Period.

115. In ignorance of the adverse facts concerning Seattle Genetics' operations which were concealed by the misrepresentations and omissions alleged herein, Lead Plaintiff and the other members of the Class purchased or otherwise acquired Seattle Genetics securities at artificially inflated prices and relied upon the price of the securities, the integrity of the market for the securities and/or upon statements disseminated by Defendants, and were damaged upon the disclosure of Defendants' wrongdoing described herein.

116. During the Class Period, Seattle Genetics' securities were traded on an active and efficient market. Lead Plaintiff and the other members of the Class, directly relying on the materially false and misleading statements described herein, and/or relying upon the integrity of the market, purchased or otherwise acquired shares of Seattle Genetics securities at prices artificially inflated by Defendants' wrongful conduct. Had Lead Plaintiff and the other members of the Class known the truth, they would not have purchased or otherwise acquired said securities, or would not have purchased or otherwise acquired them at the inflated prices that were paid. At the time of the purchases and/or acquisitions by Lead Plaintiff and the Class, the true value of Seattle Genetics' securities was substantially lower than the prices paid by Lead Plaintiff and the other members of the Class. The market price of Seattle Genetics' securities declined sharply upon public disclosure of the facts alleged herein to the injury of Lead Plaintiff and Class members.

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117. By reason of the conduct alleged herein, Seattle Genetics and the Individual Defendants knowingly or recklessly violated Section 10(b) of the Exchange Act and Rule 10b-5 promulgated thereunder.

118. As a direct and proximate result of these Defendants' wrongful conduct, Lead Plaintiff and the other Class members suffered damages in connection with their respective purchases, acquisitions and sales of the Company's securities during the Class Period upon the disclosures alleged herein. Seattle Genetics and Individual Defendants are liable for damages in connection with these losses under Section 10(b) of the Exchange Act and Rule 10b-5 promulgated thereunder.

COUNT II

Violation of § 20(a) of the Exchange Act

(against Defendants Siegall, Simpson, and Drachman)

119. Lead Plaintiff repeats and realleges allegations contained in Paragraphs 1 to 118 above, as if fully set forth herein.

120. During the Class Period, Defendants Siegall, Simpson and Drachman participated in the operation and management of Seattle Genetics, and conducted and participated, directly and indirectly, in the conduct of Seattle Genetics' business affairs. Because of their senior positions, they knew the adverse non-public information about Seattle Genetics' operations, SGN-CD33A and its known hepatotoxicity.

121. As officers of a publicly owned company, these Defendants had a duty to disseminate accurate and truthful information with respect to Seattle Genetics' reports and filings and to correct promptly any public statements issued by Seattle Genetics, which had become materially false or misleading.

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122. Because of their positions of control and authority as senior officers of the Company, Defendants Siegall, Simpson and Drachman were able to, and did, control the contents of the various reports, press releases, investor conferences and public filings which Seattle Genetics disseminated in the marketplace during the Class Period. Throughout, the Class Period, Defendants Siegall, Simpson and Drachman exercised power and authority to cause Seattle Genetics to engage in the wrongful conduct complained of herein.

123. As control persons, Defendants Siegall, Simpson and Drachman are liable pursuant to Section 20(a) of the Exchange Act for the primary violations of the Exchange Act committed by Seattle Genetics as set forth in Count I.

REQUEST FOR RELIEF

Lead Plaintiff demands judgment against Defendants as follows:

A. Determining that the instant action may be maintained as a class action under Rule 23 of the Federal Rules of Civil Procedure, and certifying Lead Plaintiff as the Class Representative;

B. Requiring Defendants to pay damages sustained by Lead Plaintiff and the Class by reason of the acts and transactions alleged herein;

C. Awarding Lead Plaintiff and the other members of the Class prejudgment and post-judgment interest, as well as their reasonable attorneys' fees, expert fees and other costs; and

D. Awarding such other and further relief as this Court may deem just and proper.

DEMAND FOR JURY TRIAL

Lead Plaintiff hereby demands a trial by jury of all issues so triable.

Dated: November 17, 2017

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Respectfully submitted,

s/ Cliff Cantor
By: Cliff Cantor, WSBA # 17893
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Plaintiffs' Lead Counsel

Certificate of Service

I certify that, on the date stamped above, I caused this document to be filed with the Clerk of the Court using the CM/ECF system, which will send notification of filing by email to counsel of record for all parties.

s/ Cliff Cantor, WSBA #17893

CERTIFICATION PURSUANT
TO FEDERAL SECURITIES LAWS

1. I, CARL JOHNSON, make this declaration pursuant to Section 27(a)(2) of the Securities Act of 1933 ("Securities Act") and/or Section 21D(a)(2) of the Securities Exchange Act of 1934 ("Exchange Act") as amended by the Private Securities Litigation Reform Act of 1995.

2. I have reviewed a Complaint against Seattle Genetics, Inc. ("Seattle Genetics" or the "Company"), and authorize the filing of a comparable complaint on my behalf.

3. I did not purchase or acquire Seattle Genetics securities at the direction of plaintiffs counsel or in order to participate in any private action arising under the Securities Act or Exchange Act.

4. I am willing to serve as a representative party on behalf of a Class of investors who purchased or acquired Seattle Genetics securities during the class period, including providing testimony at deposition and trial, if necessary. I understand that the Court has the authority to select the most adequate lead plaintiff in this action.

5. To the best of my current knowledge, the attached sheet lists all of my transactions in Seattle Genetics securities during the Class Period as specified in the Complaint.

6. During the three-year period preceding the date on which this Certification is signed, I have not sought to serve as a representative party on behalf of a class under the federal securities laws.

7. I agree not to accept any payment for serving as a representative party on behalf of the class as set forth in the Complaint, beyond my pro rata share of any recovery, except such reasonable costs and expenses directly relating to the representation of the class as ordered or approved by the Court.

8. I declare under penalty of perjury that the foregoing is true and correct.

Executed 1-9-17
(Date)

Carl Johnson
(Signature)

CARL JOHNSON
(Type or Print Name)

SEATTLE GENETICS, INC. (SGEN) **Johnson, Carl**

LIST OF PURCHASES AND SALES

DATE	PURCHASE OR SALE	NUMBER OF SHS/UTS	PRICE PER SH/UT
11/15/2016	Purchase	5,000	$71.5890
11/16/2016	Purchase	4,000	$71.2500
11/18/2016	Purchase	2,000	$72.8000
11/18/2016	Purchase	2,000	$72.1000
11/18/2016	Purchase	500	$71.1000
11/15/2016	Sale	5,000	$72.0000
11/17/2016	Sale	3,000	$72.7500
11/21/2016	Sale	100	$70.8596
11/29/2016	Sale	200	$65.8058
11/30/2016	Sale	100	$65.0326
12/8/2016	Sale	100	$65.5692
12/15/2016	Sale	100	$62.1852
12/15/2016	Sale	100	$62.1912
12/23/2016	Sale	100	$60.7440
12/23/2016	Sale	100	$60.7736

SEATTLE GENETICS, INC. (SGEN)
CLASS PERIOD: FEB 19 2016 to DEC 23 2016
(Includes 90-Day Sales @ Statutory Pricing)

Plaintiff	Purchase Date	Shares	Price	Amount	Sales Date	Shares	Price	Amount	Shares Retained	Estimated Gain(Loss)
Johnson, Carl	11/15/2016	5,000	$71.5890	($357,945)	11/15/2016	(5,000)	$72.0000	$360,000		
Johnson, Carl	11/16/2016	4,000	$71.2500	($285,000)	11/17/2016	(3,000)	$72.7500	$218,250		
Johnson, Carl	11/18/2016	2,000	$72.8000	($145,600)	11/21/2016	(100)	$70.8596	$7,086		
Johnson, Carl	11/18/2016	2,000	$72.1000	($144,200)	11/29/2016	(200)	$65.8058	$13,161		
Johnson, Carl	11/18/2016	500	$71.1000	($35,550)	11/30/2016	(100)	$65.0326	$6,503		
Johnson, Carl					12/8/2016	(100)	$65.5692	$6,557		
Johnson, Carl					12/15/2016	(100)	$62.1852	$6,219		
Johnson, Carl					12/15/2016	(100)	$62.1912	$6,219		
Johnson, Carl					12/23/2016	(100)	$60.7440	$6,074		
Johnson, Carl					12/23/2016	(100)	$60.7736	$6,077		
Johnson, Carl					12/28/2016	(2,700)	$52.7301	$142,371		
Johnson, Carl					12/29/2016	(1,900)	$53.7448	$102,115		
Johnson, Carl		**13,500**		**($968,295)**		**(13,500)**		**$880,633**	**4,800**	**($87,662)**